

**MYOCARDIAL CARNITINE DEPLETION ASSOCIATED WITH RAPID VENTRICULAR PACING.**

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Rapid ventricular pacing induces congestive heart failure (CHF) in dogs, but the mechanism of the progressive ventricular dysfunction is unclear. Carnitine is essential for fatty acid transport into mitochondria, and has been reported to be depleted in some patients with CHF. To ascertain if carnitine metabolism is altered in pacing induced CHF, we assayed myocardial carnitine and serial plasma carnitine in 11 mongrel dogs paced at 250 beats/min for 19 days. CHF was evident from reduced cardiac output ( $1.6 \pm 0.3$  L/min,  $p < 0.05$ ), lower mean arterial pressure ( $77 \pm 9$  mmHg,  $p < 0.001$ ), and elevated pulmonary wedge pressure ( $17 \pm 5$  mmHg,  $p < 0.001$ ) at 19 days, compared to control values prior to pacing of  $3.9 \pm 1.2$  L/min,  $127 \pm 30$  mmHg and  $7 \pm 3$  mmHg respectively. Plasma free carnitine (FC) and plasma total carnitine (TC) increased over control (C) as CHF progressed (\* $p < 0.05$ , \*\* $p < 0.01$ ).

|             | C          | 7 days     | 14 days         | 19 days          |
|-------------|------------|------------|-----------------|------------------|
| FC(nmol/ml) | $12 \pm 7$ | $16 \pm 8$ | $18 \pm 8^{**}$ | $24 \pm 14^{**}$ |
| TC(nmol/ml) | $15 \pm 8$ | $19 \pm 9$ | $22 \pm 9^{*}$  | $30 \pm 15^{*}$  |

Myocardial free carnitine ( $3.5 \pm 1.3$  nmol/mg non-collagenous protein) and total carnitine ( $5.6 \pm 1.5$ ) were both significantly lower ( $p < 0.001$ ) in the dogs paced for 19 days than in 8 control dogs ( $8.9 \pm 3.0$  and  $14.1 \pm 3.5$  respectively). In contrast, high energy phosphates (ATP and total adenine nucleotides) were not decreased in the CHF hearts. Thus myocardial carnitine is depleted in pacing induced CHF despite preservation of myocardial high energy phosphates and elevation of plasma carnitine. This suggests altered membrane transport of carnitine, either inadequate uptake, excessive loss, or both.

**ECHOCARDIOGRAPHIC PREDICTORS OF DEVELOPMENT OF DOUBLE-CHAMBERED RIGHT VENTRICLE IN INFANTS WITH VENTRICULAR SEPTAL DEFECT**

Pierre C. Wong, M.D., Steven D. Colan, M.D., F.A.C.C., Ira A. Parness, M.D., Stephen P. Sanders, M.D., Tal Geva, M.D., Richard Van Praagh, M.D., F.A.C.C., Philip J. Spevak, M.D. The Childrens Hospital, Boston, MA.

Double-chambered right ventricle (DCRV) is often associated with a ventricular septal defect (VSD); however, not every patient with a VSD develops DCRV. A principal mechanism of RV outflow tract obstruction in DCRV is superior displacement of the septal insertion of moderator band, and hypertrophy of the parietal band, leading to obstruction of the proximal or infundibuli. We hypothesized that it might be possible to identify these anatomic features echocardiographically in patients with VSD even before outflow tract obstruction develops, and thereby predict which patients are likely to develop DCRV. We reviewed the initial echo studies in three groups of patients: Group I (n=10), patients with VSD and DCRV; Group II (n=7), patients with VSD on the initial study but no RV outflow obstruction, who subsequently developed DCRV; Group III (n=10), an age-matched group of patients with VSD who never developed DCRV. We measured the following: distance from pulmonary valve to insertion of moderator band on the ventricular septum (PV-M); width of the parietal band; tricuspid and pulmonary valve annulus diameter; Doppler gradient across the RV outflow tract. Results (mean values  $\pm$  S.D.):

|   | Group I<br>(Known DCRV) | Group II<br>(VSD $\rightarrow$ DCRV) | Group III<br>(VSD no DCRV) |
|---|-------------------------|--------------------------------------|----------------------------|
| Mean age (mos)                                    | $18 \pm 9.6$            | $0.7 \pm 0.4^{*}$                    | $1.0 \pm 0.9$              |
| PV-M (mm)   | $14 \pm 3.6$            | $11 \pm 3.5^{\dagger}$               | $17 \pm 4.1$               |
| Indexed to: Tricuspid valve                       | $0.8 \pm 0.3$           | $0.9 \pm 0.2^{\dagger}$              | $1.6 \pm 0.2$              |
| Pulmonary valve                                   | $1.3 \pm 0.4$           | $1.3 \pm 0.3^{\dagger}$              | $2.1 \pm 0.3$              |
| Gradient (mm Hg)                                  | $56 \pm 13$             | $8.2 \pm 0.9^{\dagger}$              | $4.0 \pm 2.9$              |
| Parietal band width indexed<br>to pulmonary valve | $1.0 \pm 0.3$           | $1.0 \pm 0.2^{\dagger}$              | $0.5 \pm 0.2$              |

\* $p < 0.01$  Groups I vs II       $\dagger p < 0.01$  Groups II vs III

We conclude that the moderator band is superiorly displaced and the parietal band already thickened—even at the initial echocardiographic study—in infants with VSD who subsequently develop DCRV. These observations can be made readily by 2-D echocardiography, and might be useful in predicting which infants with VSD will eventually develop DCRV.

Monday, March 19, 1990

10:30AM-12:00NOON, Room 36

**Advances in Diagnostic Methods for Pediatric Cardiology**

FORAMEN OVALE FLOW IN THE SECOND AND THIRD TRIMESTER FETUS AND THE DIAGNOSIS OF FORAMEN OBSTRUCTION. Lisa K. Hornberger, M.D., Sarah Tabbutt, Ph.D., Viktor Chobot, M.D. Sandy Hagen-Ansert, RDMS, David J. Sahn, M.D. FACC, Univ of Calif, San Diego, CA.

We performed spectral or color flow mapping Doppler (CD) evaluations of the foramen ovale (FO) in 32 normal fetuses, 17 to 38 weeks gestational age (GA), referred for suspected fetal arrhythmia. In 20 fetuses CD demonstrated low velocity, phasic and primarily unidirectional right-to-left atrial FO flow. The maximum color flow diameter (CFD) of the FO progressively increased linearly during gestation (CFD =  $0.21GA - 0.19$  mm;  $R = 0.94$ ). For all fetuses the FO CFD was  $0.80 \pm 0.09$  (SD) of the AO diameter and  $0.77 \pm 0.09$  of main PA size. Spectral Doppler revealed biphasic systolic/diastolic flow in 15 of 23 studies and continuous, low velocity flow in the 8 others. The peak diastolic velocity of the FO flow was  $36.6 \pm 6.8$  cm/sec with a mean velocity of  $16.0 \pm 3.3$  cm/sec, which was the same for all GAs. Primary restriction of the FO was suspected in 2 other fetuses (GA 22 and 26 wks) with right heart dilatation. In both, the FO diameter fell below the 5th percentile for GA with a mean ratio to the AO diameter of 0.49. By spectral Doppler there was uniphasic right to left FO flow (120 cm/sec) in the first fetus and triphasic flow in the second, peaking at 70 cm/sec. The first fetus became hydropic at 24 weeks and died with a restrictive FO at postmortem. The second (who also had mirror image dextrocardia) survived to term delivery but had significant RA, RV and PA enlargement at birth with tachypnea and respiratory difficulty. Our data provide normal values for FO size, flow and growth and permit the evaluation of fetuses with an asymmetric four chamber view for the possible diagnosis of restrictive FO.

**MINIATURIZED HIGH FREQUENCY PHASED ARRAY DEVICES FOR HIGH RESOLUTION NEONATAL AND INTRAOPERATIVE IMAGING.**

David J. Sahn, M.D., FACC, Diana Tasker, Sandra Hagen-Ansert, RDMS, Axel Briskin, Scott Corbett. Univ of Calif, San Diego, and General Electric Co, Milwaukee, WI.

In order to optimize the quality of cardiac imaging for small neonates and improve the ease of performing high resolution echocardiography we have collaborated on the design of specialized new miniaturized phased array probes for pediatric and intraoperative use. We have produced two near-field optimized, 64-element, 7.5MHz phased array devices which have an aperture or footprint of  $0.65\text{cm}^2$ . The probes incorporate a new probe casing and an extremely flexible 68 cable connector with a diameter of 0.21 inches. The probes weigh 2 oz, and to facilitate near-field imaging they have an acoustic elevational focus between 1.5-3 cm, achieved using a special rubber facing which works best when minimal pressure is applied to the chest wall. When compared to commercially available 5MHz probes in 110 premature infants (420-2300 gms), our arrays produced very high quality imaging with good color flow and Doppler sensitivity and were extremely useful for defining pulmonary veins, the Ao arch and achieving high resolution imaging of the pulmonary valve, ductus and RV wall structures. The devices provided submillimeter lateral resolution and were easy to use even in small babies intraoperatively, providing high resolution color flow images. The near-field optimized ultrasound imaging obtained suggests applicability for transesophageal use in small babies, and a 7.5 MHz array has recently been incorporated into a 5mm pediatric endoscope. Our new imaging devices improve the quality and ease of neonatal and intraoperative ultrasound imaging.